EXHIBIT 16

W.R. Grace & Co. Bankruptcy

Supplemental Report on Asbestos and Disease Causation

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exposure to asbestos that determines the risk of developing disease[6]. The absurdity of the position that every exposure to asbestos makes a substantial contribution to the risk of disease can be seen from the following example. An individual who smoked two packs of cigarettes a day for forty years develops lung cancer and then claims that the second hand smoke he breathed in as he walked past the open door of a bar was an important factor in causing his disease. The only way to determine whether a specific exposure to asbestos was a factor in causing a claimant's disease is to conduct an explicit evaluation of the role of that asbestos exposure in causing the disease relative to the risk of developing the disease spontaneously and the additional risks imposed by other exposures, including other asbestos exposures. Such an evaluation involves estimating the additional risk imposed by the exposure at issue after taking into account the probability that the disease occurred spontaneously and the probability that other exposures, including other asbestos exposures, caused the disease. The contribution to risk made by any specific exposure will depend on how that exposure fits into the general pattern of exposure to asbestos for that individual. While such detailed information is not usually available, at the very least the total exposure, and the type and dimensions of the asbestos fibers to which exposure occurred (discussed in more detail below) need to be considered.

The Causes of Mesothelioma

As I have discussed in my original Expert Report and above, like all other cancers, mesothelioma can occur spontaneously without any exposure to environmental agents. In epidemiological studies, the fraction of mesotheliomas occurring in individuals with no history of exposure to asbestos ranges from 20 to over 80 percent (Pelnar, 1988; Peterson et al., 1984; McDonald, 1985; Spirtas, 1994; Parkin et al., 1997; Agudo et al., 2000). Mesothelioma has also been reported to occur in young children and even congenitally (Henderson 2000). Such cases are clearly not associated with asbestos exposure. The fraction of cases occurring with no discernable history of asbestos exposure is particularly high for peritoneal mesotheliomas among females. The claimants' experts are wrong in their claim that exposure to asbestos is the only known cause of mesothelioma in the United States. There is accumulating epidemiological evidence that high dose radiation for the treatment of cancer causes mesothelioma (Tward et al., 2006; Travis et al., 2005; Teta et al, 2007).

Dr. Frank claims that pericardial and testicular mesotheliomas are also caused by asbestos. In fact, epidemiological studies of these exceedingly rare tumors are extremely difficult to conduct and there is little evidence to indicate that they are caused by asbestos. It is hard to envisage how asbestos fibers could reach the testes. Pericardial and testicular mesotheliomas are probably spontaneously occurring tumors.

Asbestos Fiber Type and Disease

[6] In fact, this is an over-simplification. Cumulative exposure is only a crude measure of risk. For any carcinogen, the risk imposed by exposure depends on the temporal pattern of exposure. Thus, the risk depends on ages at start and end of exposure and the time-varying intensity of exposure. While such detailed information is rarely available for exposure to asbestos, the information should be used when available. Methods for using such information are available for mesothelioma.

APPENDIX 2

Fiber	K_M (Source)	Peto Formula
All	10^{-8} (EPA, 1986)	3.2 f/ml-yr
Chrysotile	$0.04x10^{-8}$ (B-C, 2005)	79.0 f/ml-yr
Amosite	-	-
Crocidolite	$30x10^{-8}$ (B-C, 2005)	0.1 f/ml-yr
Libby	$0.36x10^{-8}$ (McD, 2004)	8.9 f/ml-yr

Table 2: Estimates of the cumulative exposure to asbestos required to double the risk of mesothelioma, i.e., yield a RR = 2. I use the Peto formula for mesothelioma in cohorts exposed to asbestos. I assume that exposure occurs at a uniform rate over a 45 year period and I estimate the cumulative incidence 10 years after exposure stops. I assume that the background lifetime risk of mesothelioma is $3.6x10^{-4}$ after Price and Ware (2004). The Peto formula requires a potency estimate, K_M, which is shown in the second column of the table. EPA (1986) uses a potency estimate for all fibers combined. Berman and Crump (2005) provide separate potency estimates for chrysotile and amphiboles, but do not distinguish between amosite and crocidolite. The potency estimate for Libby is derived from the Libby potency estimate for lung cancer, K_L, in McDonald et al. (2004) under the assumption that $K_L/K_M = 10^6$ (EPA, 1986).

Exposure Quartiles (Mean)	Libby Observed	Chrysotile	Amosite	Crocidolite
0 - 11.7 (8.6)	1	0.02	0.65	4.71
11.7 - 25.2 (16.7)	4	0.04	1.07	7.76
25.2 - 113.8 (53.2)	3	0.09	2.56	18.51
> 113.8 (393.8)	4	0.42	11.49	83.10
TOTAL	12	0.58	15.77	114.08

Table 3: The observed number of mesotheliomas at Libby in quartiles of cumulative exposure categories as reported in table 4 of McDonald et al. (2004) compared with the expected number of mesotheliomas generated using the coefficients for pleural mesothelioma in table 8 of Hodgson and Darnton. The expected numbers were calculated using the mean cumulative exposure in each category. There are 406 subjects in this study, so that each quartile of exposure has roughly 100 subjects. The observed number of mesotheliomas is far lower than would be expected from crocidolite exposure.